Access DB# 45007

SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: Gaitene R. Gabel Examiner #: 76197 Date: 6/17/01
Art Unit: 1641 Phone Number 305-0807 Serial Number: 09/526582
Mail Box and Bldg/Room Location: 2015 Results Format Preferred (circle): PAPER DISK E-MAIL

If more than one search is submitted, please prioritize searches in order of need.

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc., if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: Method and Device for Detection of ApoA, ApoB and Ratio

Inventors (please provide full names): Judith Fitzpatrick and Regina Lenda Christopher Jones

Earliest Priority Filing Date: 3/16/99

Which 2 assay?

4

claims 1, 2, 5, 6, 8, 18, 24

highlighted search terms.

RECEIVED JUN 18 2001 (STIC)

check

Thanks!

:) Gail

STAFF USE ONLY

Searcher: M. Smith
Searcher Phone #:
Searcher Location:
Date Searcher Picked Up:
Date Completed: 7/16/01
Searcher Prep & Review Time: 60
Clerical Prep Time:
Online Time: 45

Type of Search

NA Sequence (#)
AA Sequence (#)
Structure (#)
Bibliographic
Litigation
Fulltext
Patent Family
Other

Vendors and cost where applicable

STN
Dialog 2 10 19
Questel/Orbit
Dr.Link
Lexis/Nexis
Sequence Systems
WWW/Internet
Other (specify)

PTO-1590 (1-2000)

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FILE COVERS 1947 - 16 Jul 2001  VOL 135 ISS 4
FILE LAST UPDATED: 15 Jul 2001  (20010715/ED)

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 substance identification.

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in CA from 1947 to the present.  On April 22, 2001, bibliographic
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=> d stat que
L2             1 SEA FILE=REGISTRY ("APO-, A-I LIPOPROTEINS"/CN OR "APO-, A1
                 LIPOPROTEINS"/CN)
L15           13 SEA FILE=REGISTRY ("APOLIPOPROTEIN B (GILLICHTHYS MIRABILIS
                 LIVER FRAGMENT)"/CN OR "APOLIPOPROTEIN B (HUMAN N-TERMINAL
                 FRAGMENT B17)"/CN OR "APOLIPOPROTEIN B (MESOCRICETUS AURATUS
                 STRAIN GOLDEN SYRIAN GENE APOB FRAGMENT)"/CN OR "APOLIPOPROTEIN
                  B (MONODELPHIS DOMESTICA C-TERMINAL FRAGMENT), RNA-EDITED"/CN
                 OR "APOLIPOPROTEIN B (SALMON C-TERMINAL FRAGMENT REDUCED)"/CN
                 OR "APOLIPOPROTEIN B MRNA CYTIDYLATE DEAMINASE"/CN OR "APOLIPOP
                 ROTEIN B MRNA EDITING ENZYME"/CN OR "APOLIPOPROTEIN B MRNA
                 EDITING PROTEIN (HUMAN)"/CN OR "APOLIPOPROTEIN B MRNA-EDITING
                 PROTEIN (HUMAN)"/CN OR "APOLIPOPROTEIN B RECEPTOR (HUMAN CLONE
                 PCR631 FRAGMENT)"/CN OR "APOLIPOPROTEIN B RECEPTOR (HUMAN
                 MONOCYTE-MACROPHAGE)"/CN OR "APOLIPOPROTEIN B RNA EDITING
                 DEAMINASE (HUMAN CLONE .LAMBDA.NI.1.0/.LAMBDA.NI2.2 CATALYTIC
                 SUBUNIT REDUCED)"/CN OR "APOLIPOPROTEIN B-100 (HUMAN PRECURSOR)
                 "/CN OR "APOLIPOPROTEIN B48 RECEPTOR (HUMAN PLACENTA GENE
                 APOB48R ISOFORM 2)"/CN OR "APOLIPOPROTEIN B48 RECEPTOR (HUMAN
                 THP-1 MONOCYTE-MACROPHAGE CELL GENE APOB48R)"/CN)
L18           16 SEA FILE=REGISTRY ("APOLIPOPROTEIN A-1"/CN OR "APOLIPOPROTEIN
                 A-1 (BEIJING DUCK BLOOD)"/CN OR "APOLIPOPROTEIN A-1 (GILLICHTHY
                 S SETA LIVER FRAGMENT)"/CN OR "APOLIPOPROTEIN A-1 (PSI-P700)
                 (OENOTHERA ELATA PLASTID-ENCODED GENE PSAA)"/CN OR "APOLIPOPROT
                 EIN A-1 (SPARUS AURATA LIVER)"/CN OR "APOLIPOPROTEIN A-I"/CN
                 OR "APOLIPOPROTEIN A-I (ANAS STRAIN BEIJING-DUCK LIVER)"/CN OR
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"APOLIPOPROTEIN A-I (COTURNIX COTURNIX JAPONICA LIVER PRECURSOR)"/CN OR "APOLIPOPROTEIN A-I (COTURNIX COTURNIX JAPONICA LIVER)"/CN OR "APOLIPOPROTEIN A-I (DANIO RERIO CLONE I-7 PRECURSOR)"/CN OR "APOLIPOPROTEIN A-I (EUROPEAN HEDGEHOG)"/CN OR "APOLIPOPROTEIN A-I (MESOCRICETUS AURATUS GENE APOAI)"/CN OR "APOLIPOPROTEIN A-I (MOUSE CLONE PMAI-5.0 GENE APOA-1)"/CN OR "APOLIPOPROTEIN A-I (PIG CLONE P34III)"/CN OR "APOLIPOPROTEIN A-I (TUPAIA GLIS LIVER)"/CN OR "APOLIPOPROTEIN A-I, PREPRO- (TUPAIA GLIS LIVER)"/CN OR "APOLIPOPROTEIN A-I, PRO- (TUPAIA GLIS LIVER)"/CN) OR ("APOLIPOPROTEIN AI (TREE SHREW LIVER)"/CN OR "APOLIPOPROTEIN AI, PREPRO- (TREE SHREW LIVER)"/CN OR "APOLIPOPROTEIN AI, PRO- (TREE SHREW LIVER)"/CN)

L22 139 SEA FILE=REGISTRY SALIVA/BI
L23 9652 SEA FILE=HCAPLUS L2 OR L18 OR (APOLIPOPROTEIN? OR APO)(2A)(A OR A1 OR AI OR A(W)I OR A(W)1)
L24 7738 SEA FILE=HCAPLUS L15 OR (APOLIPOPROTEIN? OR APO)(2A)B
L29 34071 SEA FILE=HCAPLUS L22 OR SALIVA?
L30 2 SEA FILE=HCAPLUS L29 AND L23 AND L24

=> d ibib abs hitrn l30 1-2

L30 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:666979 HCAPLUS
DOCUMENT NUMBER: 133:219802
TITLE: ELISA method and device for detection of **Apo A1, Apo B** in **saliva**
INVENTOR(S): Fitzpatrick, Judith; Lenda, Regina B.; Jones, Christopher L.
PATENT ASSIGNEE(S): Serex Inc., USA
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000055635	A1	20000921	WO 2000-US6810	20000316

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-124562 P 19990316
AB A method has been developed to detect the levels of **apolipoproteins A-1** and B in **saliva**, which is correlated with the levels of HDL and LDL in serum, resp. In

unstimulated **saliva**, the ratio of **Apo A** to **Apo B** is correlated with the ratio of HDL to LDL in serum. Albumin can be used to normalize the sample for diln. The high degree of correlation in combination with a simple, quick test that can be performed at the site of collection provides a coast effective, patient friendly means to monitor an individual's risk of heart disease. In the preferred embodiment, **saliva** prodn. is stimulated by means such as breath mint or tart soln. (such as lemon) and the effect of diln. controlled by ref. to albumin. In the most preferred embodiment, the assay is an ELISA assay performed using the Serex laminated strip format as described in U.S. Patent Nos. 5,710,009, 5,500,375, and 5,451, 504. These strips are advantageous since they serve as the collection and assay device.

REFERENCE COUNT: 3
REFERENCE(S):
(1) Abbott Lab; WO 9936784 A 1999 HCAPLUS
(2) Abbott Lab; WO 9936785 A 1999 HCAPLUS
(3) Cardiovascular Diagnostics Inc; WO 9419690 A 1994 HCAPLUS

L30 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1998:91257 HCAPLUS
DOCUMENT NUMBER: 128:240440
TITLE: Application of **Apo B** 3' DNA polymorphisms in forensic science practice
AUTHOR(S): Jin, Qiang; Sun, Guangyun; Wu, Meiyun
CORPORATE SOURCE: Department of Physical Evidence Examination, School of Forensic Medicine, West China University of Medical Sciences, Chengdu, 610041, Peop. Rep. China
SOURCE: Huaxi Yike Daxue Xuebao (1997), 28(2), 149-153
CODEN: HYDXET; ISSN: 0257-7712
PUBLISHER: Huaxi Yike Daxue
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Amp-FLP analyses of **Apo B** 3' VNTR locus in human blood stain, **saliva** (stain), semen, mixed stain and hair were carried out. The genotypes of **Apo B** 3' locus were detected accurately in blood stains kept at room temp. within 15 wk and at -20.degree. within 2 yr. In a series of paternity testing on 34 cases, 8 false alleged fathers were excluded, 6 of which were excluded by **Apo B** 3' locus alone or by **Apo B** 3' locus combined with other genetic markers. Three rape cases were investigated, and 2 suspects were excluded.

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> d stat que
L1 95 SEA FILE=REGISTRY APOLIPOPROTEIN?/CN
L2 1 SEA FILE=REGISTRY ("APO-, A-I LIPOPROTEINS"/CN OR "APO-, A1 LIPOPROTEINS"/CN)
L8 18609 SEA FILE=HCAPLUS L1 OR APOLIPOPROTEIN OR APO(W)(A OR B OR C OR E OR A1)
L15 13 SEA FILE=REGISTRY ("APOLIPOPROTEIN B (GILLICHTHYS MIRABILIS LIVER FRAGMENT)"/CN OR "APOLIPOPROTEIN B (HUMAN N-TERMINAL

FRAGMENT B17)"/CN OR "APOLIPOPROTEIN B (MESOCRICETUS AURATUS STRAIN GOLDEN SYRIAN GENE APOB FRAGMENT)"/CN OR "APOLIPOPROTEIN B (MONODELPHIS DOMESTICA C-TERMINAL FRAGMENT), RNA-EDITED"/CN OR "APOLIPOPROTEIN B (SALMON C-TERMINAL FRAGMENT REDUCED)"/CN OR "APOLIPOPROTEIN B MRNA CYTIDYLATE DEAMINASE"/CN OR "APOLIPOPROTEIN B MRNA EDITING ENZYME"/CN OR "APOLIPOPROTEIN B MRNA EDITING PROTEIN (HUMAN)"/CN OR "APOLIPOPROTEIN B MRNA-EDITING PROTEIN (HUMAN)"/CN OR "APOLIPOPROTEIN B RECEPTOR (HUMAN CLONE PCR631 FRAGMENT)"/CN OR "APOLIPOPROTEIN B RECEPTOR (HUMAN MONOCYTE-MACROPHAGE)"/CN OR "APOLIPOPROTEIN B RNA EDITING DEAMINASE (HUMAN CLONE .LAMBDA.NI.1.0/.LAMBDA.NI2.2 CATALYTIC SUBUNIT REDUCED)"/CN OR "APOLIPOPROTEIN B-100 (HUMAN PRECURSOR)"/CN OR "APOLIPOPROTEIN B48 RECEPTOR (HUMAN PLACENTA GENE APOB48R ISOFORM 2)"/CN OR "APOLIPOPROTEIN B48 RECEPTOR (HUMAN THP-1 MONOCYTE-MACROPHAGE CELL GENE APOB48R)"/CN)

L16 9 SEA FILE=REGISTRY ("APOLIPOPROTEIN C-I (HUMAN CLONE F19374 GENE APOC1 N-TERMINAL FRAGMENT)"/CN OR "APOLIPOPROTEIN C-II (CHICKEN CLONE T1 C-TERMINAL FRAGMENT)"/CN OR "APOLIPOPROTEIN C-II (ONCORHYNCHUS MYKISS PRECURSOR)"/CN OR "APOLIPOPROTEIN C-III (CAVIA PORCELLUS LIVER PRECURSOR)"/CN OR "APOLIPOPROTEIN C-III (HUMAN GENE APOC3 ISOENZYME APOC-III-ALA23)"/CN OR "APOLIPOPROTEIN C-III (HUMAN GENE APOC3 ISOENZYME APOC-III-THR23)"/CN OR "APOLIPOPROTEIN C2 (CATTLE FRAGMENT)"/CN OR "APOLIPOPROTEIN C2 (MOUSE CLONE MAPOC2C4 PRECURSOR)"/CN OR "APOLIPOPROTEIN CII (GALLUS DOMESTICUS CLONE T1 C-TERMINAL FRAGMENT)"/CN OR "APOLIPOPROTEIN CIII (MOUSE CLONE PMCIII-4.7 GENE APOC-3 PRECURSOR)"/CN)

L17 11 SEA FILE=REGISTRY ("APOLIPOPROTEIN E (CATTLE GENE APOE)"/CN OR "APOLIPOPROTEIN E (DANIO RERIO CLONE E1 GENE APOE PRECURSOR)"/CN OR "APOLIPOPROTEIN E (DANIO RERIO PRECURSOR)"/CN OR "APOLIPOPROTEIN E (HUMAN CLONE F19374 GENE APOE)"/CN OR "APOLIPOPROTEIN E (HUMAN GENE APOE)"/CN OR "APOLIPOPROTEIN E (ONCORHYNCHUS MYKISS CLONE T5A-54 GENE APOE PRECURSOR)"/CN OR "APOLIPOPROTEIN E (RAINBOW TROUT CLONE T5A-54 GENE APOE PRECURSOR)"/CN OR "APOLIPOPROTEIN E (SCOPHTHALMUS MAXIMUS CLONE E5 GENE APOE C-TERMINAL FRAGMENT)"/CN OR "APOLIPOPROTEIN E (SHEEP STRAIN SUFFOLK GENE APOE)"/CN OR "APOLIPOPROTEIN E (SWINE GENE APOE PRECURSOR REDUCED)"/CN OR "APOLIPOPROTEIN E (SWINE PRECURSOR)"/CN OR "APOLIPOPROTEIN E (ZEBRAFISH CLONE E1 GENE APOE PRECURSOR)"/CN OR "APOLIPOPROTEIN E RECEPTOR 2 (MOUSE GENE APOER2 PRECURSOR)"/CN OR "APOLIPOPROTEIN E RECEPTOR 2 (MOUSE GENE APOER2)"/CN OR "APOLIPOPROTEIN E3 (MOUSE)"/CN)

L18 16 SEA FILE=REGISTRY ("APOLIPOPROTEIN A-1"/CN OR "APOLIPOPROTEIN A-1 (BEIJING DUCK BLOOD)"/CN OR "APOLIPOPROTEIN A-1 (GILLICHTHYS SETA LIVER FRAGMENT)"/CN OR "APOLIPOPROTEIN A-1 (PSI-P700) (OENOTHERA ELATA PLASTID-ENCODED GENE PSAA)"/CN OR "APOLIPOPROTEIN A-1 (SPARUS AURATA LIVER)"/CN OR "APOLIPOPROTEIN A-I"/CN OR "APOLIPOPROTEIN A-I (ANAS STRAIN BEIJING-DUCK LIVER)"/CN OR "APOLIPOPROTEIN A-I (COTURNIX COTURNIX JAPONICA LIVER PRECURSOR)"/CN OR "APOLIPOPROTEIN A-I (COTURNIX COTURNIX JAPONICA LIVER)"/CN OR "APOLIPOPROTEIN A-I (DANIO RERIO CLONE I-7 PRECURSOR)"/CN OR "APOLIPOPROTEIN A-I (EUROPEAN HEDGEHOG)"/CN OR "APOLIPOPROTEIN A-I (MESOCRICETUS AURATUS GENE APOAI)"/CN OR "APOLIPOPROTEIN A-I (MOUSE CLONE PMAI-5.0 GENE APOA-1)"/CN

OR "APOLIPOPROTEIN A-I (PIG CLONE P34III)"/CN OR "APOLIPOPROTEI N A-I (TUPAIA GLIS LIVER)"/CN OR "APOLIPOPROTEIN A-I, PREPRO- (TUPAIA GLIS LIVER)"/CN OR "APOLIPOPROTEIN A-I, PRO- (TUPAIA GLIS LIVER)"/CN) OR ("APOLIPOPROTEIN AI (TREE SHREW LIVER)"/CN OR "APOLIPOPROTEIN AI, PREPRO- (TREE SHREW LIVER)"/CN OR "APOLIPOPROTEIN AI, PRO- (TREE SHREW LIVER)"/CN)

L19 6 SEA FILE=REGISTRY ("APOLIPOPROTEIN A-II (MACACA FASCICULARIS CLONE EMBL3A-II1)"/CN OR "APOLIPOPROTEIN A-II BAMP-1 (BOVINE ANTIMICROBIAL-1) (CATTLE SERUM)"/CN OR "APOLIPOPROTEIN A-IV (GALLUS DOMESTICUS)"/CN OR "APOLIPOPROTEIN A-IV (GILLICHTHYS MIRABILIS LIVER N-TERMINAL FRAGMENT)"/CN OR "APOLIPOPROTEIN A-IV (SYNTHETIC 71-AMINO ACID FRAGMENT)"/CN OR "APOLIPOPROTEIN A-IV (SYNTHETIC 84-AMINO ACID FRAGMENT)"/CN)

L22 139 SEA FILE=REGISTRY SALIVA/BI

L23 9652 SEA FILE=HCAPLUS L2 OR L18 OR (APOLIPOPROTEIN? OR APO)(2A)(A OR A1 OR AI OR A(W)I OR A(W)1)

L24 7738 SEA FILE=HCAPLUS L15 OR (APOLIPOPROTEIN? OR APO)(2A)B

L25 7421 SEA FILE=HCAPLUS L16 OR L17 OR (APOLIPOPROTEIN? OR APO)(2A)(C OR E)

L26 9653 SEA FILE=HCAPLUS L23 OR L19

L29 34071 SEA FILE=HCAPLUS L22 OR SALIVA?

L30 2 SEA FILE=HCAPLUS L29 AND L23 AND L24

L31 13 SEA FILE=HCAPLUS L29 AND (L26 OR L24 OR L25 OR L8)

L32 11 SEA FILE=HCAPLUS L31 NOT L30

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L32 ANSWER 1 OF 11 HCAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2001:396929 HCAPLUS
DOCUMENT NUMBER: 135:16375
TITLE: Oxidized apolipoproteins and methods of use
INVENTOR(S): Stocker, Roland; Wang, Xing Li; Wilcken, David
PATENT ASSIGNEE(S): The Heart Research Institute Limited, Australia
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001038395	A1	20010531	WO 2000-AU1463	20001127

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: AU 1999-4293 A 19991126

AB The present invention relates to oxidized forms of apolipoproteins and methods of use in diagnosis and treatment of diseases assocd. with oxidative stress, such as cardiovascular diseases, in particular of atherosclerosis. Specifically, oxidized forms of ApoA-I and ApoA-II having specific methionine residues oxidized are provided. Methods of use include increasing efflux of .alpha.-tocopherol (.alpha.-TOH), lowering lipid concns. and preventing or treating lipid-assocd. conditions are provided. Methods of detecting oxidative stress, genotypes and extent of lipid assocd. conditions are also provided by utilizing the oxidized apolipoproteins. ApoA of human HDL was isolated and selectively oxidized and characterized. Oxidized apoAs were detd. in human aortas. The proportion of apoA-I present as the oxidized form was substantially greater in lesions than plasma HDL. Furthermore, more oxidized apoA-I was found in late stages of atherosclerosis compared to early stage.

REFERENCE COUNT: 11

REFERENCE(S):
(1) Anantharamaiah, G; Journal of Lipid Research 1988, V29(3), P309 HCAPLUS
(3) Garner, B; The Journal of Biological Chemistry 1998, V273(11), P6080 HCAPLUS
(4) Garner, B; The Journal of Biological Chemistry 1998, V273(11), P6088 HCAPLUS
(6) Matsuura; US 5900359 A 1999 HCAPLUS
(7) Panzenbock, U; The Journal of Biological Chemistry 2000, V275(26), P19536 HCAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 2 OF 11 HCAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:781367 HCAPLUS
DOCUMENT NUMBER: 134:205669

TITLE: In a search of biological markers for Alzheimer's disease

AUTHOR(S): Couderc, R.
CORPORATE SOURCE: Service de biochimie, Hopital Trousseau, Paris, 75571/12, Fr.

SOURCE: Ann. Biol. Clin. (2000), 58(5), 581-593
CODEN: ABCLAI; ISSN: 0003-3898
PUBLISHER: John Libbey Eurotext
DOCUMENT TYPE: Journal; General Review
LANGUAGE: French

AB A review, with 58 refs. Peripheral markers for Alzheimer's disease are of interest to confirm the diagnosis, to perform epidemiol. screening, to identify distinct groups of patients, to predict the outcome of the disease, to monitor its progression and its sensibility to treatment and to give help in performing studies on the relationship between brain and behavior and on the pathophysiol. of the Alzheimer's disease. The ideal biomarker for Alzheimer's disease should detect a fundamental feature of neuropathol. and be validated in neuropathol. confirmed cases and be confirmed by at least two independent studies; should be as sensitive and specific than the clin. diagnosis (about 85% and 80%), reliable, reproducible, simple to perform, inexpensive and non invasive (studies on blood, urine, **saliva**, or buccal scrapings) or moderately invasive (skin, rectal biopsies, bone marrow samples, or cerebrospinal fluid). Such a marker has not yet been found. In this paper we present those markers which come closest to fulfilling criteria for a useful

biomarker, kipping in mind that these criteria depends on what purpose it is used (screening, prediction, diagnosis, monitoring, pathophysiol. studies...) and that the finding of a good marker depends on the understanding of the disease.

REFERENCE COUNT: 59
REFERENCE(S): (1) Alves-Rodrigues, A; Trends Neurosci 1998, V21, P516 HCAPLUS
(3) Andreasen, N; Neurosci Lett 1999, V273, P5 HCAPLUS
(6) Artiga, M; FEBS Lett 1998, V421, P105 HCAPLUS
(7) Artiga, M; Hum Mol Genet 1998, V7, P1887 HCAPLUS
(9) Baskin, F; Neurology 2000, V54, P1907 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 3 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:657385 HCAPLUS
DOCUMENT NUMBER: 134:129656
TITLE: Apoliprotein E and multiple sclerosis: impact of the epsilon-4 allele on susceptibility, clinical type and progression rate
AUTHOR(S): Hogh, Peter; Oturai, Anette; Schreiber, Karen; Blinkenberg, Morten; Jorgensen, Ole Steen; Ryder, Lars; Paulson, Olaf B.; Sorensen, Per Soelberg; Knudsen, Gitte Moos
CORPORATE SOURCE: Department of Neurology, University Hospital of Copenhagen, Copenhagen, DK-2100, Den.
SOURCE: Mult. Scler. (2000), 6(4), 226-230
CODEN: MUSCFZ; ISSN: 1352-4585
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The purpose of this study was to investigate the relation between APOE genotype and Multiple Sclerosis (MS) in a genetically homogeneous population. Two hundred-forty patients consulting the MS-clinic were examd. during a period of 3 yr (1996-1999). The mean age of the patients was 41.7 yr. As a measure of the progression rate (PR), the last registered Expanded Disability Status Scale (EDSS) score was divided by the time span (years) from disease onset until the latest assessment. The APOE genotype was detd. from **saliva** and/or blood samples using PCR-techniques. The prevalence of different APOE genotypes was compared with the allele-distribution in a population of 361 persons from a Danish cross-sectional population study. The frequency of APOE-.epsilon.4/.epsilon.4 homozygotes was higher in the MS-group as compared to controls, whereas the frequency distribution of other genotypes did not differ. The rate of progression was faster in the APOE-.epsilon.4/.epsilon.4 homozygotes compared to other genotypes in the MS group. Apparently, the APOE-.epsilon.4/.epsilon.4 homozygotes have an increased risk of developing MS. MS patients with the APOE-.epsilon.4/.epsilon.4 allele may also have an increased rate of disease progression.

REFERENCE COUNT: 35
REFERENCE(S): (3) Coraddu, F; Neurogenetics 1998, V2, P24 HCAPLUS
(4) Corder, E; Science 1993, V261, P921 HCAPLUS
(7) Ebers, G; Nature Genet 1996, V13, P472 HCAPLUS
(12) Haines, J; Hum Mol Genet 1998, V7, P1229 HCAPLUS

(14) Haines, J; Nature Genet 1996, V13, P469 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 4 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:468650 HCAPLUS
DOCUMENT NUMBER: 131:99535
TITLE: Multiplexed analysis of clinical specimens apparatus and methods
INVENTOR(S): Chandler, Van S.; Fulton, Jerrold R.; Chandler, Mark B.
PATENT ASSIGNEE(S): Luminex Corporation, USA
SOURCE: PCT Int. Appl., 301 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9936564	A1	19990722	WO 1999-US918	19990115
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9923220	A1	19990802	AU 1999-23220	19990115
PRIORITY APPLN. INFO.:			US 1998-8387	19980116
			WO 1999-US918	19990115

AB A method for the multiplexed diagnostic and genetic anal. of enzymes, DNA fragments, antibodies, and other biomols. comprises the steps of constructing an appropriately labeled beadset, exposing the beadset to a clin. sample, and analyzing the combined sample/beadset by flow cytometry is disclosed. Flow cytometric measurements are used to classify, in real-time, beads within an exposed beadset and textual explanations, based on the accumulated data obtained during real-time anal., are generated for the user. The inventive technol. enables the simultaneous, and automated, detection and interpretation of multiple biomols. or DNA sequences in real-time while also reducing the cost of performing diagnostic and genetic assays.

REFERENCE COUNT: 16
REFERENCE(S): (4) Davis; US 5853984 A 1998 HCAPLUS
(5) Jutila; US 5756095 A 1998 HCAPLUS
(9) Saunders; US 4665020 A 1987 HCAPLUS
(10) Schwartz; US 4714682 A 1987 HCAPLUS
(12) Vlieger, A; Analytical Biochemistry 1992, V205(1), P1 HCAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 5 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:184150 HCAPLUS

DOCUMENT NUMBER: 130:205169
TITLE: Method for treating a subject suffering from conditions associated with an extracellular zinc sphingomyelinase
INVENTOR(S): Tabas, Ira; Schissel, Scott L.; Williams, Kevin Jon
PATENT ASSIGNEE(S): The Trustees of Colombia University in the City of New York, USA
SOURCE: PCT Int. Appl., 189 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9911283	A1	19990311	WO 1998-US18362	19980904
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 5989803	A	19991123	US 1997-937234	19970908
AU 9893016	A1	19990322	AU 1998-93016	19980904
EP 1009426	A1	20000621	EP 1998-945870	19980904
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1997-937234 A2 19970905
WO 1998-US18362 W 19980904

AB The present invention provides for a method for treating a subject suffering from a condition assocd. with an extracellular zinc sphingomyelinase activity which comprises administering to the subject an amt. of a zinc sphingomyelinase inhibitor effective to decrease extracellular zinc sphingomyelinase activity in the subject and thereby treat the subject. The present invention also provides for a method for detg. whether a compd. inhibits an activity of an extracellular zinc sphingomyelinase involving ceramide formation which comprises: (a) contacting a sample contg. the zinc sphingomyelinase under acidic pH conditions known to be assocd. with the activity of such zinc sphingomyelinase, with: (i) a substrate of the zinc sphingomyelinase enzyme, and (ii) the compd. being evaluated; (b) measuring the concn. of ceramide in the sample from (a); (c) detg. the amt. of zinc sphingomyelinase activity in the sample based upon the concn. of ceramide measured in step (b); and (d) comparing the amt. of sphingomyelinase activity detd. in step (c) with the amt. of sphingomyelinase activity detd. in the absence of the compd., to det. whether the compd. inhibits the activity of zinc sphingomyelinase.

REFERENCE COUNT: 8
REFERENCE(S):
(1) Catsimpoolas; US 4766111 A 1988 HCAPLUS
(2) Ito; JP 8113535 A 1996
(3) Murakami; JP 6211668 A 1994

(4) Nara; JP 7258132 A 1995
(5) Nara; JP 8134002 A 1996
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 6 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:543568 HCAPLUS
DOCUMENT NUMBER: 127:158939
TITLE: Bacteriocalcifins: calcium binding proteolipids of Corynebacterium matruchotii and the genes encoding them and the prevention and treatment of calcification-related disease
INVENTOR(S): Boyan, Barbara D.; Van Dijk, Simon; Dean, David D.
PATENT ASSIGNEE(S): Board of Regents, University of Texas System, USA; Boyan, Barbara D.; Van Dijk, Simon; Dean, David D.
SOURCE: PCT Int. Appl., 115 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9725430	A1	19970717	WO 1997-US528	19970110
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2242430	AA	19970717	CA 1997-2242430	19970110
AU 9715781	A1	19970801	AU 1997-15781	19970110
AU 728185	B2	20010104		
EP 873408	A1	19981028	EP 1997-902012	19970110
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 5989907	A	19991123	US 1997-780836	19970110
JP 2000505645	T2	20000516	JP 1997-525458	19970110

PRIORITY APPLN. INFO.: US 1996-9798 P 19960111
WO 1997-US528 W 19970110

AB A new class of proteolipids, called bacteriocalcifins, of Corynebacterium matruchotii that induce in vitro calcium binding are described and the genes encoding them are cloned and characterized. The proteins may play a role in dental plaque formation and in heart valve calcification. A proteolipid was extd. from cultures of C. matruchotii and sepd. into a lipid component and three apoproteins having mol. wts. of approx. 5.0, 5.5 and 7.5 kDa as detd. by SDS-PAGE. The 5.5 kDa protein shows sequence similarities to mammalian phosphoprotein phosphatases but there are no similarities to calcification proteins of Streptococcus. The invention also includes the polyclonal and monoclonal antibodies directed against the membrane assocd. proteolipid and immunol. assays developed with these antibodies.

IT **193487-79-5P**
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
(amino acid sequence; bacteriocalcifin calcium binding proteolipids of Corynebacterium matruchotii and genes encoding them and prevention and treatment of calcification-related disease)

L32 ANSWER 7 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:361636 HCAPLUS
DOCUMENT NUMBER: 126:327752
TITLE: Multiplexed analysis of clinical specimens apparatus and method
INVENTOR(S): Chandler, Van S.; Fulton, R. Jerrold; Chandler, Mark B.
PATENT ASSIGNEE(S): Luminex Corporation, USA; Chandler, Van S.; Fulton, R. Jerrold; Chandler, Mark B.
SOURCE: PCT Int. Appl., 293 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9714028	A2	19970417	WO 1996-US16198	19961010
W: AL, AM, AT, AU, BA, BB, BG, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA				
US 5736330	A	19980407	US 1995-542401	19951011
US 5981180	A	19991109	US 1995-540814	19951011
CA 2227895	AA	19970417	CA 1996-2227895	19961010
AU 9673989	A1	19970430	AU 1996-73989	19961010
EP 852004	A2	19980708	EP 1996-936310	19961010
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI				
US 6057107	A	20000502	US 1998-55329	19980406

PRIORITY APPLN. INFO.:
US 1995-540814 19951011
US 1995-542401 19951011
WO 1996-US16198 19961010

AB A method for the multiplexed diagnostic and genetic anal. of enzymes, DNA fragments, antibodies, and other biomols. comprises the steps of constructing an appropriately labeled bead set, exposing the bead set to a clin. sample, and analyzing the combined sample/bead set by flow cytometry. Flow cytometric measurements are used to classify, in real-time, beads within an exposed bead set and textual explanations, based on the accumulated data obtained during real-time anal., are generated for the user. The inventive technol. enables the simultaneous,

and automated, detection and interpretation of multiple biomols. or DNA sequences in real-time while also reducing the cost of performing diagnostic and genetic assays.

L32 ANSWER 8 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:658649 HCAPLUS
DOCUMENT NUMBER: 125:294510
TITLE: Molecular cloning of the mouse **apolipoprotein** D gene and its upregulated expression in Niemann-Pick disease type C mouse model
AUTHOR(S): Yoshida, Kunihiro; Cleaveland, Emily S.; Nagle, James W.; French, Stefanie; Yaswen, Linda; Ohshima, Toshio; Brady, Roscoe O.; Pentchev, Peter G.; Kulkarni, Ashok B.
CORPORATE SOURCE: Gene Targeting Research Core Facility, National Institute Dental Research, Bethesda, MD, 20892, USA
SOURCE: DNA Cell Biol. (1996), 15(10), 873-882
CODEN: DCEBE8; ISSN: 1044-5498
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Apolipoprotein** D (ApoD) is a member of the lipocalin superfamily. The primary structure and diverse expression of ApoD suggest that this protein is a multiligand, multifunctional glycoprotein. Here the authors report the structure of the mouse ApoD gene, which is composed of six exons spanning approx. 20 kb in length. All the exon-intron splice junctions follow the consensus GT/AG sequence. The 5'-flanking region of the mouse ApoD gene contains several putative regulatory elements, including FSE-2, GRE, SDR, MRE, IL-6RE, and TATA box. Northern blot anal. revealed that ApoD was highly expressed in the brain and adipose tissue in mouse. Lower levels of expression were obsd. in the heart, lung, thymus, testis, and **salivary** glands. In situ hybridization for the brain showed that ApoD mRNA was mainly localized in the subarachnoid space including the pia. In the Niemann-Pick disease type C mouse model, ApoD expression was upregulated in many organs such as brain, adipose tissue, heart, and thymus, presumably due to enhanced ApoD synthesis in perivascular fibroblasts.

L32 ANSWER 9 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1994:262540 HCAPLUS
DOCUMENT NUMBER: 120:262540
TITLE: Mouse **apolipoprotein** J: characterization of a gene implicated in atherosclerosis
AUTHOR(S): Jordan-Starck, Tuajuanda C.; Lund, S. Diane; Witte, David P.; Aronow, Bruce J.; Ley, Catherine A.; Stuart, William D.; Swertfeger, Debi K.; Clayton, Lisa R.; Sells, Stephen F.; et al.
CORPORATE SOURCE: Coll. Med., Univ. Cincinnati, Cincinnati, OH, 45267, USA
SOURCE: J. Lipid Res. (1994), 35(2), 194-210
CODEN: JLPRAW; ISSN: 0022-2275
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Apolipoprotein** J (apoJ), **a** glycoprotein assocd. with subclasses of plasma high d. lipoproteins (HDL), was found to accumulate

in aortic lesions in a human subject with transplantation-assocd. arteriosclerosis and in mice fed a high-fat atherogenic diet. Foam cells present in mouse aortic valve lesions expressed apoJ mRNA, suggesting local synthesis contributes to apoJ's localization in atherosclerotic plaque. As a prerequisite for elucidating the physiol. function of apoJ by using a mouse model, cDNA clones representing the mouse homolog of apoJ were isolated, characterized, and sequenced. The nucleotide sequence predicts a 448 amino acid, 50,260 dalton protein. Their was 81% nucleotide sequence similarity between mouse and human apoJ, and 75% similarity at the amino acid level. Mouse apoJ contains six potential N-glycosylation sites, a potential Arg-Ser cleavage site to generate .alpha. and .beta. subunits, a cluster of five cysteine residues in each subunit, three putative amphipathic helixes, and four potential heparin-binding domains. Southern blot anal. indicates that the gene encompasses .apprx.23 kb of DNA. Recombinant inbred strains were used to map apoJ to mouse chromosome 14, tightly linked to Mtv-11. All of the transcribed portions of the gene were cloned and analyzed, and all intron-exon boundaries were defined. The first of the 9 exons is untranslated. Single exons encode the signal peptide, the cysteine-rich domain in the .alpha. subunit, two potential amphipathic helixes flanking a heparin-binding consensus sequence and a potential amphipathic helix overlapping a heparin-binding domain, supporting their potential functional significance in apoJ. A variety of mouse tissues constitutively express a 1.9 kb apoJ mRNA, with apparently identical transcriptional start sites utilized in all tissues tested. ApoJ mRNA was most abundant in stomach, liver, brain, and testis, with intermediate levels in heart, ovary, and kidney. The high degree of similarity between mouse and human apoJ, in structure and distribution of the gene product, gene structure, and deposition in atherosclerotic plaques, suggests that the mouse is an ideal model with which to elucidate the role of apoJ in HDL metab. and atherogenesis.

IT **154609-89-9, Apolipoprotein** J (Mouse clone .lambda.dash48 gene apoJ .alpha.-chain) **154609-90-2, Apolipoprotein** J (Mouse clone .lambda.dash48 gene apoJ .beta.-chain)
RL: PRP (Properties)
(amino acid sequence and domains of)

L32 ANSWER 10 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1990:566752 HCAPLUS
DOCUMENT NUMBER: 113:166752
TITLE: The uses of computer-aided signal peptide selection and polymerase chain reaction in gene construction and expression of secreted proteins
AUTHOR(S): Daugherty, Bruce L.; Zavodny, Susan M.; Lenny, Albert B.; Jacobson, Marlene A.; Ellis, Ronald W.; Law, Simon W.; Mark, George E.
CORPORATE SOURCE: Dep. Cell. Mol. Biol., Merck, Sharp and Dohme Res. Lab., Rahway, NJ, 07065, USA
SOURCE: DNA Cell Biol. (1990), 9(6), 453-9
CODEN: DCEBE8; ISSN: 1044-5498
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The computer program, SIGSEQ2, was used to select heterologous signal

peptides from a catalog of published sequences to express the echistatin gene in insect (Sf9) cells. S-values for each amino acid were detd. to select empirically the site of cleavage between the signal peptide and mature echistatin. Five gene fragments encoding the signal peptides for human Ig kappa (Ig.kappa.), Drosophila 68C glue, antistasin, bovine growth hormone (bGH), and human **apolipoprotein E** (**Apo E**) were constructed by the use of long synthetic oligonucleotides or polymerase chain reaction (PCR). Echistatin expression vectors then were constructed using the baculovirus polyhedrin promoter. Following transient transfection, the media were assayed for echistatin activity. The results indicate that the computer program greatly facilitated the selection and design of 5 different signal peptides and accurately predicted their relative functionality in the expression and secretion of echistatin in insect cell cultures.

L32 ANSWER 11 OF 11 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1984:508033 HCAPLUS
DOCUMENT NUMBER: 101:108033
TITLE: Pattern of serum protein gene expression in mouse visceral yolk sac and fetal liver
AUTHOR(S): Meehan, Richard R.; Barlow, Denise P.; Hill, Robert E.; Hogan, Brigid L. M.; Hastie, Nicholas D.
CORPORATE SOURCE: MRC Clin. Popul. Cytogenet. Unit, West. Gen. Hosp., Edinburgh, EH4 2XU, UK
SOURCE: EMBO J. (1984), 3(8), 1881-5
CODEN: EMJODG; ISSN: 0261-4189
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mol. hybridization has shown that the mRNAs for albumin, transferrin, **apolipoprotein-A1**, and .alpha.1-antitrypsin are expressed at high levels in mouse visceral yolk sac. In contrast, the mRNAs for contrapsin (a plasma protease inhibitor) and the major urinary proteins (MUPs) are not detected in the visceral yolk sac at any stage of embryonic development. Contrapsin and MUP mRNAs both appear late in liver development. These differences in expression suggest that the visceral yolk sac is more similar to the fetal than adult mouse liver in its pattern of gene expression. However, the developmental time course of expression of these mRNAs is different between the fetal liver and the yolk sac. Evidence is also presented that the visceral yolk sac synthesizes and secretes other apolipoproteins in addn. to **apolipoprotein-A1**. Apparently the visceral yolk sac and fetal liver, 2 tissues with different embryol. lineages, perform similar functions but are independently programmed for expression of the same set of serum protein genes.

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NO E#s ASSIGNED
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E1 THROUGH E3 ASSIGNED

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DICTIONARY FILE UPDATES:  15 JUL 2001  HIGHEST RN 345949-33-9

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               (154609-89-9/RN)
             1 154609-90-2/BI
               (154609-90-2/RN)
             1 193487-79-5/BI
               (193487-79-5/RN)
L33          3 (154609-89-9/BI OR 154609-90-2/BI OR 193487-79-5/BI)

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L33  ANSWER 1 OF 3  REGISTRY  COPYRIGHT 2001 ACS
RN   193487-79-5  REGISTRY
CN   L-Leucine, L-methionyl-L-.alpha.-aspartyl-L-tyrosylglycyl-L-glutaminyl-L-
     isoleucyl-L-alanyl-L-.alpha.-glutamyl-L-glutaminyl-L-leucylglycyl-L-
     asparaginyl-L-phenylalanyl-L-lysyl-L-lysyl-L-phenylalanyl-L-alanyl-L-
     .alpha.-glutamyl-L-alanyl-L-isoleucylglycylglycyl-L-isoleucyl-L-
     phenylalanyl-L-threonyl-L-.alpha.-glutamyl-L-leucyl-L-prolyl-L-lysyl-L-
     phenylalanyl-L-leucyl-L-asparaginyl-L-asparaginyl-L-leucyl-L-.alpha.-
     aspartyl-L-seryl-L-phenylalanyl-L-valylglycylglycylglycyl-L-arginylglycyl-
     L-seryl-L-seryl-L-.alpha.-glutamyl- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Apolipoprotein (Corynebacterium matruchotii strain ATCC-14266
     calcium-precipitating 5.0-kilodalton)
CN   Bacteriocalcifin (Corynebacterium matruchotii)
CN   Proteolipid, apo- (Corynebacterium matruchotii strain ATCC-14266
     calcium-precipitating 5.0-kilodalton)
FS   PROTEIN SEQUENCE
DR   209051-90-1
MF   C229 H350 N58 O70 S
CI   MAN
SR   CA
LC   STN Files:   CA, CAPLUS, USPATFULL

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M. Smith 308-3278

2 REFERENCES IN FILE CA (1967 TO DATE)
2 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 129:64432

REFERENCE 2: 127:158939

=> d ide can 2-3

L33 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2001 ACS
RN **154609-90-2** REGISTRY
CN Lipoprotein NA 1 (mouse clone .lambda.dash48 .beta.-chain reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Apolipoprotein J (Mouse clone .lambda.dash48 gene apoJ .beta.-chain)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 120:262540

L33 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2001 ACS
RN **154609-89-9** REGISTRY
CN Lipoprotein NA 1 (mouse clone .lambda.dash48 .alpha.-chain reduced) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Apolipoprotein J (Mouse clone .lambda.dash48 gene apoJ .alpha.-chain)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXLIT

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 120:262540

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         (c) 2001 Inst for Sci Info
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         (c) 2001 CAB International
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         (c) 2001  Elsevier Science B.V.
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         (c) 2001 Elsevier Science B.V.
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         (c) 2001 The Gale Group
File 151:HealthSTAR 1975-2000/Dec
         (c) format only 2000 The Dialog Corporation
File 185:Zoological Record Online(R) 1978-2001/Jun
         (c) 2001 BIOSIS
File 342:Derwent Patents Citation Indx 1978-01/200135
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File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
         (c) 1998 Inst for Sci Info
File 440:Current Contents Search(R) 1990-2001/Jul W4
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013439596
WPI Acc No: 2000-611539/200058
XRAM Acc No: C00-183001
XRPX Acc No: N00-452877
 Immunodetection of  apolipoproteins in  saliva is useful for monitoring
 risk of heart disease in an individual
Patent Assignee: SEREX INC (SERE-N)
Inventor: FITZPATRICK J; JONES C L; LENDA R B
Number of Countries: 089  Number of Patents: 002
Patent Family:
Patent No      Kind   Date     Applicat No     Kind   Date      Week
WO 200055635    A1  20000921  WO 2000US6810    A   20000316  200058  B
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AU 200038861 A 20001004 AU 200038861 A 20000316 200101

Priority Applications (No Type Date): US 99124562 A 19990316
Patent Details:
Patent No Kind Lan Pg Main IPC Filing Notes
WO 200055635 A1 E 34 G01N-033/92
Designated States (National): AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
Designated States (Regional): AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
AU 200038861 A G01N-033/92 Based on patent WO 200055635

Abstract (Basic): WO 200055635 A1
Abstract (Basic):
NOVELTY - Detection of an apolipoprotein comprising reacting saliva with antibodies immunoreactive with the apolipoprotein is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) an assay device or kit for the novel method, comprising means for collection of saliva and antibodies immunoreactive with an apolipoprotein ; and
(2) quantitating the amount of lipoprotein or cholesterol in saliva or the presence of lipid disorders or risk of cardiovasular disease, comprising reacting a saliva sample with antibodies specifically immunoreactive with apolipoprotein selected from ApoA , ApoB , ApoC , ApoE or components of them.
USE - The method and assay devices/kits are useful for detection of apolipoprotein in saliva , which is correlated with levels of high-density lipoprotein and low-density lipoprotein in serum. The method and assay devices/kits are useful for quantitating the amount of lipoprotein or cholesterol in saliva or the presence of lipid disorders or risk of cardiovascular disease (claimed).
ADVANTAGE - The high degree of correlation of the ratio of Apo A to Apo B and the ratio of high density lipoprotein (HDL) to low density lipoprotein (LDL) in combination with a simple, quick test that can be performed at the site of collection provides a cost effective, patient friendly means to monitor an individual's risk of heart disease.
pp; 34 DwgNo 0/4
?ds

Set	Items	Description
S1	909	(SALIVA OR SPUTUM) AND (APOLIPOPROTEIN? OR APO?)
S2	406	RD (unique items)
S3	361	(SALIVA OR SPUTUM) (S) (APOLIPOPROTEIN? OR APO?)
S4	1	S3 AND MUCOPOLYSACC?
S5	10	S3 AND (HDL OR LDL OR CHOLESTEROL OR LIPOPROTEIN?)
S6	9	S5 NOT S4

?t s6/3 ab/1-9
>>>No matching display code(s) found in file(s): 342

6/AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07121814 94195460 PMID: 8145881
[Macromolecular inhibitors of crystallization in saliva and bile]
Les inhibiteurs macromoleculaires de cristallisation dans la salive et dans la bile.

Verdier JM
INSERM U315, Unite de Recherches et Pathologie Digestives, Marseille.
Nephrologie (SWITZERLAND) 1993, 14 (6) p251-5, ISSN 0250-4960
Journal Code: NX4
Languages: FRENCH
Document type: Journal Article; Review; Review, Tutorial
Record type: Completed
The current knowledges concerning macromolecular inhibitors of crystallization in saliva and in bile are reviewed. In saliva , four families of inhibiting proteins have been evidenced: the statherins, the acidic proline-rich proteins, the cystatins and the histatins. These proteins inhibit the nucleation and the growth of calcium phosphate salts. In the bile, two families of proteins that inhibit the nucleation of calcium carbonate and cholesterol are present: the apolipoproteins and the calcium binding protein also called anionic polypeptide fraction. The structure-function relationships of these molecules are particularly stressed.

6/AB/2 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03005338 Genuine Article#: MX496 Number of References: 0
(NO REFS KEYED)
Title: MACROMOLECULAR INHIBITORS OF CRYSTALLIZATION IN BILE AND SALIVA (Abstract Available)
Author(s): VERDIER JM
Corporate Source: INSERM,U315,UNITE RECH & PATHOL DIGEST,46 BLVD GAYE/F-13009 MARSEILLE//FRANCE/
Journal: NEPHROLOGIE, 1993, V14, N6, P251-255
ISSN: 0250-4960
Language: FRENCH Document Type: ARTICLE
Abstract: The current knowledges concerning macromolecular inhibitors of crystallization in saliva and in bile are reviewed. In saliva , four families of inhibiting proteins have been evidenced: the statherins, the acidic proline-rich proteins, the cystatins and the histatins. These proteins inhibit the nucleation and the growth of calcium phosphate salts. In the bile, two families of proteins that inhibit the nucleation of calcium carbonate and cholesterol are present: the apolipoproteins and the calcium binding protein also called anionic polypeptide fraction. The structure-function relationships of these molecules are particularly stressed.

6/AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

05678472 EMBASE No: 1994081392
Macromolecular inhibitors of crystallization in bile and saliva
LES INHIBITEURS MACROMOLECULAIRES DE CRISTALLISATION DANS LA SALIVE ET DANS LA BILE
Verdier J.-M.
INSERM U315, Unite de Rech./Pathologie Digestives, 46 Boulevard de la Gaye,F-13009 Marseille France
Nephrologie (NEPHROLOGIE) (Switzerland) 1993, 14/6 (251-255)
CODEN: NEPHD ISSN: 0250-4960
DOCUMENT TYPE: Journal; Short Survey
LANGUAGE: FRENCH SUMMARY LANGUAGE: ENGLISH; FRENCH

The current knowledge concerning macromolecular inhibitors of crystallization in saliva and in bile are reviewed. In saliva , four families of inhibiting proteins have been evidenced: the statherins, the acidic proline-rich proteins, the cystatins and the histatins. These proteins inhibit the nucleation and the growth of calcium phosphate salts. In the bile, two families of proteins that inhibit the nucleation of calcium carbonate and cholesterol are present: the apolipoproteins and the calcium binding protein also called anionic polypeptide fraction. The structure-function relationships of these molecules are particularly stressed.

6/AB/4 (Item 1 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01675305 SUPPLIER NUMBER: 19175111 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Diagnosing pulmonary alveolar proteinosis: a review and an update.
Wang, Bennet M.; Stern, Eric J.; Schmidt, Rodney A.; Pierson, David J.
Chest, v111, n2, p460(7)
Feb,
1997
PUBLICATION FORMAT: Magazine/Journal ISSN: 0012-3692 LANGUAGE: English
RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 4050 LINE COUNT: 00352

6/AB/5 (Item 2 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01619731 SUPPLIER NUMBER: 18306605 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Cystic fibrosis in adults: from researcher to practitioner.
Marelich, Gregory P.; Cross, Carroll E.
The Western Journal of Medicine, v164, n4, p321(14)
April,
1996
PUBLICATION FORMAT: Magazine/Journal ISSN: 0093-0415 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 13175 LINE COUNT: 01133

AUTHOR ABSTRACT: The Cystic Fibrosis Foundation currently tracks about 20,000 Americans with cystic fibrosis, an autosomal recessive genetic disease that leads to multisystem complications. With the institution of better therapeutic regimens over the past 2 decades, more patients with this disease are surviving to adulthood. Within the past decade, both clinical and basic science research in the field of cystic fibrosis has progressed at a rapid rate. The intent of this review is to introduce readers to the molecular, cellular, and systemic disorders of this disease. We discuss treatment strategies involving antibiotics, nutrition, immune-response mediators, chest physiotherapy, and sputum-active agents with respect to the airway dysfunction of cystic fibrosis. Other common complications, recent developments, transplantation, and gene therapy are also reviewed.

6/AB/6 (Item 3 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01414561 SUPPLIER NUMBER: 13635193 (USE FORMAT 7 OR 9 FOR FULL TEXT)

Surfactant protein-A concentration in bronchoalveolar lavage fluids of patients with pulmonary alveolar proteinosis.
Honda, Yasuhito; Takahashi, Hiroki; Shijubo, Noriharu; Kuroki, Yoshio; Akino, Toyoaki
Chest, v103, n2, p496(4)
Feb,
1993
PUBLICATION FORMAT: Magazine/Journal ISSN: 0012-3692 LANGUAGE: English
RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 2329 LINE COUNT: 00247

6/AB/7 (Item 1 from file: 342)
DIALOG(R)File 342:Derwent Patents Citation Indx

04230836 WPI Acc No: 00-611539/58
Immunodetection of apolipoproteins in saliva is useful for monitoring risk of heart disease in an individual -
Patent Assignee: (SERE-) SEREX INC
Author (Inventor): FITZPATRICK J; LENDA R B; JONES C L
Patent (basic)

Patent No	Kind	Date	Examiner	Field of Search
WO 200055635	A1	000921 (BASIC)		

Derwent Week (Basic): 0058
Priority Data: US 124562 (990316)
Applications: AU 200038861 (000316); WO 2000US6810 (000316)
Designated States
(National): AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CR; CU; CZ; DE; DK; DM; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW
(Regional): AT; BE; CH; CY; DE; DK; EA; ES; FI; FR; GB; GH; GM; GR; IE; IT; KE; LS; LU; MC; MW; NL; OA; PT; SD; SE; SL; SZ; TZ; UG; ZW
Derwent Class: B04; D16; J04; S03
Int Pat Class: G01N-033/92
Number of Patents: 002
Number of Countries: 089
Number of Cited Patents: 003
Number of Cited Literature References: 000
Number of Citing Patents: 000

6/AB/8 (Item 1 from file: 351)
DIALOG(R)File 351:Derwent WPI

013871697
WPI Acc No: 2001-355909/200137
XRAM Acc No: C01-110431
Novel oxidized form of apolipoprotein useful in diagnosis and treatment of diseases associated with oxidative stress such as cardiovascular diseases, in particular, atherosclerosis
Patent Assignee: HEART RES INST LTD (HEAR-N)
Inventor: STOCKER R; WANG X L; WILCKEN D
Number of Countries: 094 Number of Patents: 001
Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Week	
WO 200138395	A1	20010531	WO 2000AU1463	A	20001127	200137	B

Priority Applications (No Type Date): AU 994293 A 19991126
Patent Details:
Patent No Kind Lan Pg Main IPC Filing Notes
WO 200138395 A1 E 55 C07K-014/775
Designated States (National): AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
Designated States (Regional): AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

Abstract (Basic): WO 200138395 A1
Abstract (Basic):

NOVELTY - An oxidized apolipoprotein , apoA-I (I) with at least a Met residue 86 oxidized to Met (O), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an oxidized apoA-II (Ia) comprising an apoA-II dimer which is oxidized at a single or several Met residue(s);

(2) a reconstituted high density lipoprotein (HDL) (II) comprising (I);

(3) assessing (M1) genotype, by detecting in a biological sample the presence of an oxidized apolipoprotein with an oxidation at a single or several Met residue(s) and comparing against a condition where the genotype is absent or different;

(4) diagnosing a lipid-associated condition is characterized by high lipid concentration and/or genetic factors known to be associated with an increased risk of cardiovascular disease, by determining the levels of an apolipoprotein oxidized at a single or several Met residue(s) and comparing against levels in an absence of the lipid-associated condition;

(5) determining the extend of a lipid-associated condition characterized by a high lipid and/or apolipoprotein concentration adjusted level of an oxidized apolipoprotein with an oxidation at a single or several Met residue(s) and comparing against an absence of the lipid-associated condition;

(6) measuring oxidative stress in vivo, by detecting the presence of an oxidized apolipoprotein with an oxidation at a single or several Met residue(s) and comparing against a condition in the absence of oxidative stress; and

(7) collecting biological samples for determination of oxidized apolipoproteins , by obtaining a biological sample and subjecting the sample to conditions which reduce further oxidation of a single or several methionine residue(s) of the apolipoprotein .

ACTIVITY - Cardiant; antiarteriosclerotic; vasotropic; antilipemic.

MECHANISM OF ACTION - Inducer of alpha-tocopherol and lipid efflux from cells (claimed).

To determine the ability of apoA-I to promote cellular lipid efflux, the rate of clearance of multilamelar DMPC liposomes by lipid-free apoA-I and apoA-I+32 was examined. The kinetics of liposome clearance were similar, although the time required for initial relative turbidity ((A0-A.)/A0) to decrease to half (i.e., t1/2) was significantly shorter for apoA-I+32 than apoA-I. Thus, the rate constant, k1/2(k1/2=1/t1/2) (28) was 0.7+/-0.4 and 1.6+/-0.8 minute-1 for apoA-I and apoA-I+32 respectively (mean+/-SD, n=9, P=0.0003). The resulting ratio of k1/2-A-I+32/k1/2-A-I was 2.4+/-0.5, indicating that apoA-I+32 converted multilamellar liposomes to small unilamellar vesicles 2-3 times faster than apoA-I. The result suggested that the introduction of the sulfoxide groups increased the ability of apoA-I on interact with phospholipids.

USE - (I) or (Ia) is useful for inducing an increased efflux of lipids from cells, inducing an increased efflux of alpha-tocopherol (alpha-TOH) from cells, lowering lipid concentration in cells e.g. monocyte derived macrophages, and preventing or treating lipid-associated conditions e.g. coronary vascular disease, ischemic heart disease, atherosclerosis and dyslipidemias, where the condition is associated with high levels of lipids (claimed). The genotype to be assessed is associated with an increased risk of coronary disease for detecting an increased risk of coronary disease, a polymorphism of a genotype associated with an increased risk for coronary disease, or associated with an increased risk of cardiovascular disease, selected from endothelial nitric oxide synthase (eNOS) genotype a/b, Asp298 variant of eNOS, or conditions resulting in high levels of circulating homocysteine (claimed).

ADVANTAGE - The oxidized lipoproteins provide indicators of diseases which are associated with oxidative stress to give a better diagnosis of heart conditions and cardiovascular disease.

pp; 55 DwgNo 0/8

6/AB/9 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

05227469 GENUINE ARTICLE#: MX496 NUMBER OF REFERENCES: 0
(NO REFS KEYED)
TITLE: MACROMOLECULAR INHIBITORS OF CRYSTALLIZATION IN BILE AND SALIVA
AUTHOR(S): VERDIER JM
CORPORATE SOURCE: INSERM,U315,UNITE RECH & PATHOL DIGEST,46 BLVD GAYE/F-13009 MARSEILLE//FRANCE/ (Reprint)
PUBLICATION: NEPHROLOGIE, 1993, V14, N6, P251-255
ISSN: 0250-4960
LANGUAGE: FRENCH DOCUMENT TYPE: ARTICLE
ABSTRACT: The current knowledges concerning macromolecular inhibitors of crystallization in saliva and in bile are reviewed. In saliva , four families of inhibiting proteins have been evidenced: the statherins, the acidic proline-rich proteins, the cystatins and the histatins. These proteins inhibit the nucleation and the growth of calcium phosphate salts. In the bile, two families of proteins that inhibit the nucleation of calcium carbonate and cholesterol are present: the apolipoproteins and the calcium binding protein also called anionic polypeptide fraction. The structure-function relationships of these molecules are particularly stressed.

?ds

Set	Items	Description
S1	909	(SALIVA OR SPUTUM) AND (APOLIPOPROTEIN? OR APO?)
S2	406	RD (unique items)
S3	361	(SALIVA OR SPUTUM) (S) (APOLIPOPROTEIN? OR APO?)
S4	1	S3 AND MUCOPOLYSACC?
S5	10	S3 AND (HDL OR LDL OR CHOLESTEROL OR LIPOPROTEIN?)
S6	9	S5 NOT S4
S7	132	S3 AND (ANTIBOD? OR AB OR MAB? ? OR PAB? ? OR AB? ? OR IMMUNO?)
S8	72	S3 (S) (ANTIBOD? OR AB OR MAB? ? OR PAB? ? OR AB? ? OR IMMUNOR?)
S9	23	RD (unique items)
S10	19	S9 NOT S5
S11	13	S10 NOT APOPTO?

?t s11/3 ab/1-13
>>>No matching display code(s) found in file(s): 342

11/AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10919893 20480064 PMID: 11023828
Macromolecular organization of saliva: identification of 'insoluble' MUC5B assemblies and non-mucin proteins in the gel phase.
Wickstrom C; Christersson C; Davies JR; Carlstedt I
Mucosal Biology Group, Department of Cell and Molecular Biology, Section for Molecular Pathogenesis, Lund University, P.O. Box 94, S-22100 Lund, Sweden.
Biochemical journal (ENGLAND) Oct 15 2000, 351 Pt 2 p421-8, ISSN 0264-6021 Journal Code: 9YO
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Stimulated human submandibular/sublingual (HSMSL) and whole saliva were separated into sol and gel phases and mucins were isolated by density-gradient centrifugation in CsCl/4M guanidinium chloride. MUC5B and MUC7 were identified using anti-peptide antisera raised against sequences within the MUC5B and MUC7 apoproteins respectively. MUC7 was found mainly in the sol phase of both HSMSL and whole saliva , but some MUC7 was consistently present in the gel phase, suggesting that this mucin may interact with the salivary gel matrix. In HSMSL saliva , MUC5B was found in the gel phase; however, most of the material was 'insoluble' in guanidinium chloride and was only brought into solution by reduction. In whole saliva , the MUC5B mucin was present both in the sol and gel phases although some material was again 'insoluble'. Rate-zonal centrifugation of whole saliva showed that MUC5B mucins in the sol phase were smaller than those in the gel phase, suggesting differences in oligomerization and/or degradation. Antibodies against IgA, secretory component, lysozyme and lactoferrin were used to study the distribution of non-gel-forming proteins in the different phases of saliva . The majority of these proteins was found in the sol phase of both HSMSL and whole saliva . However, a significant fraction was present in the gel phase of whole saliva , suggesting a post-secretory interaction with the salivary gel matrix. A monoclonal antibody against a parotid salivary agglutinin was used to show that this protein is present mainly in the gel phase of both whole saliva and parotid secretion.

11/AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09151948 97135427 PMID: 8980964
Surfactant apoprotein-A concentration in airway secretions for the detection of pulmonary oedema.
Shimura S; Masuda T; Takishima T; Shirato K
First Department of Internal Medicine, Tohoku University School of Medicine, Sendai, Japan.
European respiratory journal (DENMARK) Dec 1996, 9 (12) p2525-30, ISSN 0903-1936 Journal Code: ERY
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Patients with cardiogenic pulmonary oedema expectorate foamy sputum containing surfactant, which might be expected to include surfactant apoprotein A (SP-A). SP-A is specific for lung surfactant. We have measured the SP-A concentration in airway secretions to determine whether

it is useful in distinguishing pulmonary oedema from other disorders. Samples of sputum and of aspirated airway secretion were obtained from 11 patients with cardiogenic pulmonary oedema, seven patients with clinically stable congestive heart failure, five patients with adult respiratory distress syndrome (ARDS) and 20 control patients (10 intubated) with other respiratory diseases. The samples were used for the measurement of SP-A concentration by a two-site simultaneous immunoassay with monoclonal antibodies against SP-A. SP-A concentrations, measured in samples of sputum and aspirated secretions, depended on the diagnosis of the patients from which they had come. In descending order these samples came from patients with: cardiogenic pulmonary oedema (1324 +/- 197 micrograms.mL-1; n = 33); ARDS (311 +/- 47 micrograms.mL-1; n = 23); clinically stable congestive heart failure (78 +/- 10 micrograms.mL-1; n = 21); and control conditions (3:0 +/- 0.6 micrograms.mL-1; n = 30). Concentrations from disease samples did not overlap with controls. In samples from patients with cardiogenic pulmonary oedema, the SP-A concentration correlated with mean pulmonary capillary wedge pressure (PCWP) ($p < 0.001$; n = 39). These findings indicate that the measurement of the surfactant apoprotein A concentration in airway secretions may be useful for the detection of pulmonary oedema.

11/AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08465371 95145638 PMID: 7843330
Proteinaceous precursors of human axillary odor: isolation of two novel odor-binding proteins.
Spielman AI; Zeng XN; Leyden JJ; Preti G
New York University, College of Dentistry, Division of Basic Sciences, New York.
Experientia (SWITZERLAND) Jan 15 1995, 51 (1) p40-7, ISSN 0014-4754 Journal Code: EQZ
Contract/Grant No.: DC-01072, DC, NIDCD
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The characteristic odor which arises in the human axillary region consists of volatile C6-C11 acids with the most abundant being (E)-3-methyl-2-hexenoic acid (E-3M2H). This acid, as well as several other components of the characteristic axillary odor, can be liberated from the odorless, aqueous soluble components of apocrine secretion by either saponification or bacteriolysis. It is therefore likely that a major characteristic odor is being carried to the skin surface bound to a water soluble precursor where it is liberated by axillary bacteria. The individual proteins found in apocrine secretions were separated, isolated and hydrolyzed with the resultant hydrolyzates analyzed by gas chromatography/mass spectrometry. These studies demonstrated that 3M2H was liberated from 2 proteins with apparent molecular mass of 26 and 45 kilodaltons: Apocrine Secretion Odor-Binding Protein 1 and 2, respectively (ASOB1 and ASOB2). Antisera to these proteins were prepared and used to examine a variety of other body fluids. Several fluids contained an immunoreactive protein with the same electropheretic migration pattern as the 45 KDa protein. Three of these body fluids (tears, nasal secretions and saliva) were separated into aqueous and organic soluble fractions and hydrolyzed to demonstrate that 3M2H could be liberated from the aqueous soluble materials. These results suggest interesting parallels between non-human mammalian odors used as chemical signals and human axillary odor. Previous studies have suggested the axillae as a source of human primer-type pheromones; consequently, if the

odors which characterize the underarm are responsible for the pheromonal activity, then the chemistry involved may be similar to that in other mammalian chemical communication systems where proteins act as carriers of one or more chemical signals.

11/AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07550055 91155569 PMID: 1671943
Surfactant apoprotein-A concentration in sputum for diagnosis of pulmonary alveolar proteinosis.
Masuda T; Shimura S; Sasaki H; Takishima T
First Department of Internal Medicine, Tohoku University School of Medicine, Sendai, Japan.
Lancet (ENGLAND) Mar 9 1991, 337 (8741) p580-2, ISSN 0140-6736 Journal Code: L0S
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Pulmonary alveolar proteinosis (PAP), a disease characterised by accumulation of surfactant in alveoli, is diagnosed on the basis of invasive biopsy procedures. We have measured apoprotein A (SP-A) concentrations in sputum to see if this is useful for the diagnosis of PAP. Sputum samples from three patients with PAP and twenty patients with other pulmonary disease were assayed using monoclonal antibodies to SP-A. SP-A concentrations were 400 times higher in patients with PAP than in the controls, suggestions that this measurement is useful for the diagnosis of PAP especially where lung biopsy is contraindicated.

11/AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06534146 88182055 PMID: 2895603
Haemorrhagic fever with renal syndrome: clinical, virological and epidemiological perspectives.
Chan YC; Wong TW; Yap EH
Department of Microbiology, Faculty of Medicine, National University of Singapore.
Annals of the Academy of Medicine, Singapore (SINGAPORE) Oct 1987, 16 (4) p696-701, ISSN 0304-4602 Journal Code: 53F
Languages: ENGLISH
Document type: Journal Article; Review; Review, Tutorial
Record type: Completed
Haemorrhagic fever with renal syndrome (HFRS) is caused by a group of RNA viruses within the family of Bunyaviridae known as hantaviruses. The classical, severe form of HFRS is characterized by fever, headache, abdominal and lumbar pain, proteinuria, haemorrhagic phenomena, shock and renal failure. The disease is associated with the prototype Hantaan virus and occurs in rural areas of Korea and China with Apodemus mice as reservoir hosts. A clinically less severe form of HFRS, which is caused by Seoul virus, occurs in urban areas with the house rat Rattus novegicus as the main reservoir host. The disease in nonendemic areas may be atypical and patients with symptoms the hepatitis and minimal renal involvement have been observed in Malaysia. Outbreaks of HFRS in humans involving infected laboratory rat colonies have occurred in several medical centres in various countries. Hantaviruses cause a chronic, asymptomatic infection in rodents which excrete the virus in their lungs, saliva and urine. Man becomes

infected mainly by inhalation of infected droplets from healthy rodent carriers. Seroepidemiological studies using mainly the indirect immunoflourescent antibody test of sera from humans and rats showed that hantaviruses have a worldwide distribution.

11/AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05812852 86158339 PMID: 2869749
Pathogenesis of experimental Hantaan virus infection in laboratory rats.
Lee PW; Yanagihara R; Gibbs CJ; Gajdusek DC
Archives of virology (AUSTRIA) 1986, 88 (1-2) p57-66, ISSN 0304-8608 Journal Code: 8L7
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Weanling Fischer rats inoculated intramuscularly with Hantaan virus (strain 76-118) developed subclinical infections characterized by transient viremia and shedding of virus in saliva , persistence of virus in lung, pancreas, spleen and liver, and development of fluorescent and neutralizing antibodies in serum with immune complex deposition in lung. Viremia and virus shedding in saliva occurred 10 to 13 days after inoculation. Horizontal intracage transmission of infection occurred between 35 and 63 days post-inoculation, long after disappearance of virus in oropharyngeal secretions and blood. Multiple attempts to demonstrate infectious virus in feces and urine during this period were unsuccessful. The inability to detect virus in urine samples of experimentally infected rats may have resulted from intermittent or low-titered viruria. This contrasts sharply with the prolonged high-titered viruria reported in striped field mice (Apodemus agrarius) infected with Hantaan virus, suggesting differences in the mode(s) of virus transmission in nature.

11/AB/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

03997764 82021597 PMID: 6116436
Intraspecific transmission of Hantaan virus, etiologic agent of Korean hemorrhagic fever, in the rodent Apodemus agrarius.
Lee HW; Lee PW; Baek LJ; Song CK; Seong IW
American journal of tropical medicine and hygiene (UNITED STATES) Sep 1981, 30 (5) p1106-12, ISSN 0002-9637 Journal Code: 3ZQ
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Experimental parameters of infection and intraspecific transmission of Hantaan virus, the etiologic agent of Korean hemorrhagic fever, in Apodemus agrarius rodents were determined. Mice inoculated by the intramuscular route experienced viremia for about 1 week beginning on day 7. After 3 weeks, immunofluorescent and neutralizing antibodies were present and no mouse ever developed signs of acute illness. Virus was recovered from lung, kidney, salivary gland, and liver, and virus excretion in urine, saliva , and feces occurred from about day 10 through day 360 (urine) post-inoculation. Antigen, but not infectious virus, was persistent in lung tissue for as long as 1 year. Horizontal contact infection occurred among cage-mates regardless of sexual pairing up to 360 days after infection and no evidence for participation of ectoparasitic arthropods in such transmission was obtained.

11/AB/8 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

05047037 BIOSIS NO.: 000081005161
STUDY OF HORIZONTAL AND VERTICAL TRANSMISSION OF HANTAAN VIRUS IN APODEMUS-AGRARIUS AND RATS
AUTHOR: PARK K-K
AUTHOR ADDRESS: DEP. MICROBIOL., COLL. MED., KOREA UNIV., SEOUL, KOREA.
JOURNAL: KOREA UNIV MED J 22 (1). 1985. 33-42. 1985
FULL JOURNAL NAME: Korea University Medical Journal
CODEN: CUIHA
RECORD TYPE: Abstract
LANGUAGE: KOREAN

ABSTRACT: It was reported that Hantaan virus is excreted in urine and saliva of A. agarius infected with the virus and ectoparasites do not play an important role in transmission of Hantaan virus among Apodemus mice. There is no report on horizontal inter-cage transmission of the virus among Apodemus mice in the ordinary animal room nor vertical transmission of the virus in rodents. Horizontal contact infection occurred among cage-mates regardless of sexual paring. Inter-cage transmission of Hantaan virus was demonstrated between Apodemus mice that were kept 1-4 m apart in an animal room. Infection of normal mice with the virus from infected mice that were kept in other cages occurred after a 2 mo. in the animal room where many infected Apodemus mice were kept. Vertical transmission of Hantaan virus in the infected pregnant Apodemus mice caught in the endemic areas of HFRS (hemorrhagic fever with renal syndrome) and the infected Apodemus mice inoculated with the virus was not demonstrated. Vertical transmission of Hantaan virus in the pregnant Wistar rats inoculated with the virus was not observed, but passage of IF antibodies to the virus through placenta of infected mother rats to baby rats was demonstrated.

1985

11/AB/9 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

13298525 PASCAL No.: 98-0021753
The antifungal effect of lactoferrin and lysozyme on Candida krusei and Candida albicans
SAMARANAYAKE Y H; SAMARANAYAKE L P; WU P C; SO M
Faculty of Dentistry, The University of Hong Kong, Hong Kong; Department of Pathology, Faculty of Medicine, The University of Hong Kong, Hong Kong; Department of Statistics, The University of Hong Kong, Hong Kong
Journal: APMIS. Acta pathologica, microbiologica et immunologica Scandinavica, 1997, 105 (11) 875-883
Language: English
Lactoferrin and lysozyme (muramidase) are non-immune defence factors present in various exocrine secretions, including saliva . Previous studies have shown that both proteins, either singly or in combination, are bactericidal in nature and their combined activity is synergistic. As little is known of their interactions with Candida species, 20 oral isolates of C. krusei and 5 isolates of C. abi .arts were studied for their susceptibility to human apo -lactoferrin and lysozyme, either singly or in combination, using an in vitro assay system. The two species

exhibited significant interspecies differences in susceptibility to lactoferrin ($p<0.05$), but not for lysozyme; C. krusei being more sensitive to lactoferrin (c 1.4 times) than C. abicans. Both species revealed significant intraspecies differences in their susceptibility to lysozyme ($p<0.05$), but not for lactoferrin. No synergistic antifungal activity of the two proteins on either Can(ida species was noted. The results imply that the variable expression of the fungicidal activity of lactoferrin and lysozyme on Candida species may modulate the oral carriage of yeasts in a complex manner.

11/AB/10 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal

12100913 PASCAL No.: 95-0330201
Rice cationic peroxidase accumulates in xylem vessels during incompatible interactions with Xanthomonas aryzae pv oryzae
YOUNG S A; AILAN GUO; GUIKEMA J A; WHITE F F; LEACH J E
Kansas State univ., dep. plant pathology, Manhattan KS 66506-5502, USA
Journal: Plant physiology : (Bethesda), 1995, 107 (4) 1333-1341
Language: English
A cationic peroxidase, PO-C1 (molecular mass 42 kD, isoelectric point 8.6), which is induced in incompatible interactions between the vascular pathogen Xanthomonas orycae pv orycae and rice (Oryca saliva L.), was purified. Amino acid sequences from chemically cleaved fragments of PO-C1 exhibited a high percentage of identity with deduced sequences of peroxidases from rice, barley, and wheat. Polyclonal antibodies were raised to an 11-amino acid oligopeptide (POC1a) that was derived from a domain where the sequence of the cationic peroxidase diverged from other known peroxidases. The anti-POC1a antibodies reacted only with a protein of the same mobility as PO-C1 in extracellular and guttation fluids from plants undergoing incompatible responses collected at 24 h after infection. In the compatible responses, the antibodies did not detect PO-C1 until 48 h after infection. Immunoelectron microscopy was used to demonstrate that PO-C1 accumulated within the apoplast of mesophyll cells and within the cell walls and vessel lumen of xylem elements of plants undergoing incompatible interactions

11/AB/11 (Item 1 from file: 351)
DIALOG(R)File 351:Derwent WPI

009145652
WPI Acc No: 1992-273091/199233
XRAM Acc No: C92-121451
XRPX Acc No: N92-208873
Immunoassay of tumour markers in living body sample - by reacting substance binding specifically with components fixed on carrier and labelled components
Patent Assignee: KONICA CORP (KONS)
Number of Countries: 001 Number of Patents: 001
Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Week	
JP 4186161	A	19920702	JP 90314108	A	19901121	199233	B

Priority Applications (No Type Date): JP 90314108 A 19901121
Patent Details:

Patent No Kind Lan Pg Main IPC Filing Notes
JP 4186161 A 6 G01N-033/543

Abstract (Basic): JP 4186161 A

The method comprises reacting a sample consisting of many given components substances binding specifically with respective given components fixed on the same carrier and labelled matters of the substances.

Pref. concn. of each labelled matter is regulated based on the cut-off (threshold) value of each given component. The concn. of each labelled matter is regulated by multivariable analysis based on each determination value. Sample is e.g. blood, blood plasma, blood serum, spinal fluid, saliva , milk, urine or sweat. Given components to be determined by the method are e.g. polypeptide, protein, polysaccharide, lipid, nucleic acid, hormone, vitamin, drug or antibiotic. Substances binding specifically with the given components are e.g. antibody , antigen, lectin or protein A. Labelling substance is e.g. radioactive isotope, enzyme, substrate, apoenzyme or fluorescent substance pref. enzyme such as beta-galactosidase, alkaline phosphatase or peroxidase. Reaction type in the immunoassay is e.g. competitive method, two antibody method or sandwich method, pref. sandwich method. Insoluble carrier is e.g. agarose, cellulose, crosslinked dextran or polyacrylamide.

USE/ADVANTAGE -The method determines immunologically tumour markers esp. in living body sample. Combination assay can be simply carried out at low cost, consequently diagnosis can be accurately carried out. Accurate diagnosis can be carried out in the determn. of tumour markers

Dwg.0/0

11/AB/12 (Item 2 from file: 351)
DIALOG(R)File 351:Derwent WPI

009145649
WPI Acc No: 1992-273088/199233
XRAM Acc No: C92-121448
XRPX Acc No: N92-208870
Immunoassay for determn. of minor component in e.g. blood - comprises reacting fine insol. particles bound with (labelled) antigen or antibody, sepg. reaction prods. and determining labelling substances etc.
Patent Assignee: KONICA CORP (KONS)
Number of Countries: 001 Number of Patents: 001
Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Week	
JP 4186158	A	19920702	JP 90314105	A	19901121	199233	B

Priority Applications (No Type Date): JP 90314105 A 19901121
Patent Details:
Patent No Kind Lan Pg Main IPC Filing Notes
JP 4186158 A 13 G01N-033/543

Abstract (Basic): JP 4186158 A

Determining given component in sample by heterogeneous immunoassay comprises reacting fine insol. particles bound with antibody (or antigen) antigen (or antibody) in the sample and labelled antibody (or antigen), sepg. the reaction prods. bound with the insol. fine particles and unreacted substances, and determining the labelling substance in the unreacted substances not bound with the insol. fine particles to analyse the antigen (or antibody) in the sample.

Sample is e.g. blood, blood plasma, blood serum, spinal fluid, saliva , milk, urine, or sweat. Given component to be determined by the method, is e.g. polypeptide, protein, polysaccharide, lipid, nucleic acid, hormone, vitamin, drug or antibiotic. Labelling substance is e.g. radioactive isotope, enzyme, substrate, apoenzyme or fluorescent substance, pref. enzyme such as beta-galactosidase, alkaline phosphatase or peroxidase. Reaction type in the immunoassay is e.g. competitive, two antibody or sandwich method. Insol. carrier (fine particles) for fixing antibody (or antigen) is pref. e.g. agarose, crosslinked agarose, crosslinked dextran or polyacrylamide. The particle size of the carrier fine particles is less than 2mm, pref. 0.1-300 microns.

USE/ADVANTAGE - Dry analytical element used contains pref. at least a porous layer. Used for determining minor component in a fluid sample, esp. given minor component in biological fluid sample. Given component in fluid sample can be simply and accurately determined in a short time

Dwg.0/0

11/AB/13 (Item 3 from file: 351)
DIALOG(R)File 351:Derwent WPI

007369728
WPI Acc No: 1988-003663/198801
XRAM Acc No: C88-001770
XRPX Acc No: N88-002527
Immunoassay analytical element for sepg. fluid components - comprises porous reaction layers contg. immobilised binding reagents, labels and label enhancers etc.
Patent Assignee: KONISHIROKU PHOTO IND CO LTD (KONS)
Number of Countries: 001 Number of Patents: 001
Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Week	
JP 62267667	A	19871120	JP 86112365	A	19860515	198801	B

Priority Applications (No Type Date): JP 86112365 A 19860515
Patent Details:

Patent No	Kind	Lan	Pg	Main IPC	Filing Notes
JP 62267667	A		23		

Abstract (Basic): JP 62267667 A

An immunoassay element has at least one porous reaction layer for determining given component A in fluid sample by using substance B binding specifically with the given component A or its analogues. Substance C which binds specifically with substance B but does not bind with the given component A. Substance E binds specifically with labelling substance D to modulate a signal arising from the labelling substance D are immobilised on a carrier and the immobilised matter is contained in at least a layer of the porous reaction layers.

Fluid sample is blood, blood plasma, blood serum, spinal fluid, saliva , urine, sweat, etc. Given component to be determined by the element is polypeptide, protein, complex protein, polysaccharide, lipid, nucleic acid, hormone, vitamin, drug, antibiotic, etc. Substance B and substance C are antigen, antibody , lectin, protein A, inhibitor of enzyme, etc. The specific binding reaction of given component A and substance B is pref. antigen-antibody reaction. Antibody used is IgG, IgM, IgA, IgD, IgE, etc. Labelling substance D is enzyme, enzyme substrate, inhibitor of enzyme, apoenzyme , fluorescent substance, etc. Substance E is e.g., inhibitor of the enzyme when labelling

substance D is an enzyme. Porous reaction layer is made of granular matter of 10-350 microns or fibre of 40-400 mesh, such as granular matter of synthetic resin, diatom earth, titanium dioxide, etc. or fibre of cotton, pulp, polypropylene, etc.

ADVANTAGE - B/F sepn. is carried out in the analytical element, and given component in a fluid sample can be determined with little background and little noise by the analytical element. The sensitivity, accuracy and reproducibility by the analytical element are excellent.

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L11 ANSWER 34 OF 61 MEDLINE DUPLICATE 22
ACCESSION NUMBER: 91216022 MEDLINE
DOCUMENT NUMBER: 91216022 PubMed ID: 2022176
TITLE: Salivary secretion of albumin in type 1 (insulin-dependent) diabetes.
AUTHOR: Fisher B M; Lamey P J; Sweeney D; Beeley J A; Spooner R J; Frier B M
CORPORATE SOURCE: Diabetic Department, Western Infirmary, Glasgow Dental Hospital and School, U.K.
SOURCE: DIABETES RESEARCH AND CLINICAL PRACTICE, (1991 Feb) 11 (2) 117-9.
Journal code: EBI; 8508335. ISSN: 0168-8227.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199106
ENTRY DATE: Entered STN: 19910623
Last Updated on STN: 19910623
Entered Medline: 19910603

AB The concentration of **albumin** in **saliva** is low in healthy humans. To determine whether alterations in capillary permeability in diabetes affects the salivary glands, the concentration of **albumin** in parotid **saliva** was measured in 26 Type 1 (insulin-dependent) diabetic patients, and compared to 32 non-diabetic control subjects. The diabetic patients were subdivided into 3 groups on the basis of the urinary excretion of **albumin** in timed overnight collections of urine: (1) normal **albumin** excretion (less than 30 micrograms/min) n = 13; (2) microalbuminuria (30-300 micrograms/min) n = 7, and (3) macroalbuminuria (greater than 300 micrograms/min) n = 6. **Saliva** was collected for one minute following **stimulation** with 1 ml 10% citric acid, and the concentration of **albumin** was measured by a sensitive ELISA method. No significant difference in salivary **albumin** concentration was found between the control group and any of the diabetic groups. Thus, although urinary **albumin** excretion was increased, suggesting altered capillary permeability, simultaneous leakage of **albumin** into **saliva** was not observed. Measurement of salivary **albumin** concentration does not, therefore, provide a marker of occult microvascular disease in diabetes.

L19 ANSWER 3 OF 15 MEDLINE

ACCESSION NUMBER: 88298988 MEDLINE
DOCUMENT NUMBER: 88298988 PubMed ID: 2969903
TITLE: A solid phase enzyme immunoassay for the measurement of urinary albumin and the detection of microalbuminuria.
AUTHOR: Coppo R; Amore A; Roccatello D; Formica M; Beltrame G; Malavasi F; Sena L M; Piccoli G
CORPORATE SOURCE: Department of Medical Nephrology, University of Turin, Italy.
SOURCE: JOURNAL OF DIABETIC COMPLICATIONS, (1987 Apr-Jun) 1 (2) 58-60.
Journal code: HNO; 8708656. ISSN: 0891-6632.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198809
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880915

AB A test for the measurement of trace urinary albumin concentrations, which is suitable for the detection of microalbuminuria, was developed. The technique is an indirect enzyme-linked assay (**ELISA**) in which a fixed amount of **anti-albumin antibody** is placed into polystyrene tubes coated with human albumin, together with the urine sample to be tested. The albumin in the test specimen competes with the solid-phase albumin for binding to the added antibody. The test is precise (inter- and intra-assay coefficients of variation were 8.2% and 7.8%, respectively), accurate (mean recovery 102-106% for two human albumin preparations), and sensitive (detection limit 0.9 micrograms/ml). These characteristics are not dissimilar from those of the radioimmunoassay reported in the literature, with the advantages of being completely safe, easy to perform, and not requiring expensive equipment. Using this assay the urinary albumin excretion in 20 normal subjects was found to be 2.5 +/- 2.2 micrograms/min (range 0.9-7.5 micrograms/min) after 8 hours of bed rest and 4.5 +/- 5.7 micrograms/min (range 1.5-2.0 micrograms/min) after 8 hours of moderate physical activity.